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20. ABSTRACT (Continue on reverse side if necessary and identify by block number)

Twenty-four heat acclimatized male volunteers were wrapped as previously described but to produce miliaria rubra (heat rash) in specific regions of the body. Three experimental rash groups were involved: 1) the torso (17% total skin surface rashed, n=6), 2) torso and arms (38%, n=8), or 3) legs (41%, n=6), while four subjects served as controls. All subjects were re-exposed to walking in the heat on the 7th day after unwrapping, and again 14, 21 and 28 days after unwrapping. When compared to responses for the last heat acclimatization day,

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tolerance time and sweat rate were significantly lower and mean body temperature and Δ heat storage significantly higher for experimental rash subjects contrasted to the controls for up to 21 days; however, no significant differences between the three rashed groups were found. The critical amount of surface area for heat intolerance from heat rash appears to be related to the specific region of the body and associated sweating responses; smaller rashed areas of the trunk, because they have greater potential for abundant sweating, may produce similar responses to heat stress as larger rashed areas of the limbs. Heat intolerance due to rash was not resolved until after 21 days.

HEAT INTOLERANCE AS A FUNCTION OF PERCENT OF BODY SURFACE

INVOLVED WITH MILIARIA RUBRA

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Running head: HEAT INTOLERANCE AND INVOLVED BODY SURFACE WITH
                HEAT RASH .

Twenty-four heat acclimatized male volunteers were wrapped as previously described but to produce miliaria rubra (heat rash) in specific regions of the body. Three experimental rash groups were involved: 1) the torso (17% total skin surface rashed, n=6), 2) torso and arms (38%, n=8), or 3) legs (41%, n=6), while four subjects served as controls. All subjects were re-exposed to walking in the heat on the 7th day after unwrapping, and again 14, 21 and 28 days after unwrapping. When compared to responses for the last heat acclimatization day, tolerance time and sweat rate were significantly lower and mean body temperature and heat storage significantly higher for experimental rash subjects contrasted to the controls for up to 21 days; however, no significant differences between the three rashed groups were found. The critical amount of surface area for heat intolerance from heat rash appears to be related to the specific region of the body and associated sweating responses; smaller rashed areas of the trunk, because they have greater potential for abundant sweating, may produce similar responses to heat stress as larger rashed areas of the limbs. Heat intolerance due to rash was not resolved until after 21 days.

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Index terms: heat rash; performance time; core temperature; skin temperature; mean body temperature; heart rate; sweat rate; body heat storage; heat acclimatization; exercise; regional distribution of sweat rate; anhidrosis.

Previous work has shown that one bout of artificially induced miliaria rubra (heat rash) resulted in a marked reduction in performance and an increase in several physiological responses of these rashed individuals compared to matched controls (10). Reduced performance and elevated physiological responses of the rashed group were seen to persist for at least 14 days. The impaired heat tolerance of the rashed individuals was associated with a fatigue or failure of the sweat glands over the rashed surface.

The classic treatise on sweating is that written by Kuno (7). Sweating was shown to be most abundant on the head, the neck and trunk, and less on the extremities (7). The number of sweat glands in various parts of the body per unit surface area have been evaluated by a number of authors (7,11). However, an evaluation of the amount of sweat secreted from different body areas displays only a rough correlation between sweat output and the number of glands (7). Thus, while the percent of the total surface area involved is an important consideration for sweating function, the particular area of the body may also be an important consideration.

The initial heat rash investigation clearly demonstrated heat intolerance after "clinical recovery" from heat rash involving between 40 and 70% (mean 57%) of the subject's surface area (10). The same protocol used in the initial investigation was utilized in this study, but with involved areas of less than 50% of the subject's surface area. The post-rash evaluation was continued beyond 14 days to 28 days. The purpose of the study was to determine the critical surface area of heat rash to produce subsequent intolerance. In particular, the percent of the total surface area rashed relative to the known abundance for sweating in this area was evaluated. Also, the persistence (up to 28 days) of such intolerance associated with recovery from artificially induced heat rash was studied.

METHODS

The general protocol and experimental methodology has been described previously (10) but a brief summary and specific differences from the earlier study are given here. Twenty-four healthy, Caucasian male volunteers served as test subjects after giving their written informed consent. Initially, these twenty-four men were concurrently heat acclimatized by walking (1.56 m s^{-1}) for 7 days on a level treadmill for 100 min in an environmentally controlled chamber at a temperature (T_a) of 48.9°C and relative humidity of 20% (27.8°C, T_{wb}) with a wind velocity of 1.4 m s^{-1}.

After heat acclimatization, the twenty-four volunteers were carefully matched, according to final heart rate and final rectal temperature, and organized into four groups. Three of these groups had experimental rashing of different areas of the body (torso, legs, or torso and arms) while a control group was also evaluated. The torso rash group (21% surface covered) were wrapped (polyethylene film) as in a hip length shirt without sleeves. The leg rash group (44% surface covered) were wrapped as with trousers, with a fly front and a rear trap door. The torso-arms rash group (39% surface covered) were wrapped as in a hip length shirt with long sleeves. The torso group (n=6) had an average age (mean $\pm$SE) of 20.3 ± 0.2 yr; height, 175.2 ± 2.1 cm; weight (nude), 72.5 ± 3.4 kg, and body surface area of 1.88 ± 0.05 m^2; the legs group (n=6) had an average age of 21.8 ± 0.6 yr; height, 177.4 ± 1.8 cm; weight, 74.7 ± 1.6 kg, and body surface area of 1.92 ± 0.02 m^2; the torso-arms group (n=8) had a mean age of 21.5 ± 0.5 yr; height, 172.1 ± 1.6 cm; weight, 72.0 ± 2.6 kg, and body surface area of 1.85 ± 0.04 m^2, while the control subjects (n=4) had an average age of 23.5 ± 1.0 yr; height, 177.8 ± 2.1 cm; weight, 73.3 ± 2.7 kg, and body surface area of 1.91 ± 0.04 m^2. The method for inducing

miliaria rubra and evaluating the degree and distribution of rash was as described previously (1).

Seven days after the removal of the wrapping, all men were subjected to a repetition of the treadmill walking and heat stress test under conditions identical to the 7-day acclimatization period. This same evaluation was conducted 14, 21 and 28 days after the removal of the wraps. The physiological measures, collection times, and method of calculation were identical to those in the initial study (10).

Statistical Analysis

The same mixed factorial analysis of variance design (9) utilized previously (10) was again used in this investigation. The experimental variables evaluated were mean body temperature ($1/3 \ \overline{T}_{sk}$ plus $2/3 \ T_{re}$), heat storage, heart rate, sweat rate and tolerance time. Once statistical significance ($P < 0.05$) was established, critical differences were calculated (8) to find where the actual mean differences existed.

RESULTS

After heat acclimatization, the experimental and control groups were formed in a similar fashion to the initial study. The experimental and control groups had average (mean $\pm$ SE) values for final rectal temperature (T_{re}) and final heart rate (HR) of $39.14 \pm 0.11^\circ C$ and 151 ± 7 bts/min (legs group), $39.10 \pm 0.13^\circ C$ and 154 ± 5 bts/min (torso group), $39.14 \pm 0.15^\circ C$ and 153 ± 4 bts/min (torso and arms group), and $39.07^\circ C \pm 0.20$ and 139 ± 11 bts/min (control group). None of these differences in physiological responses between groups were statistically significant ($P > 0.05$). When the physical characteristics (Methods) were contrasted between the control group and the combined rashed groups, the only significant difference was in age,

the control group being 2 1/4 years older. These comparisons illustrate the physiological and physical similarity between groups prior to the induction of experimental rashing.

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Table 1 presents a comparison by rashed body areas of the total areas wrapped, effectively rashed, and the percent of the total body surface area rashed. Of the original total area wrapped (% of total surface area) for the legs group (44%), the torso group (21%) and torso and arms group, (39%), the average percent of total area rashed was 40.5% for the legs group, 16.7% for the torso group and 37.5% for the torso and arms group. Thus, the most effectively rashed group (rashed/wrapped, %) was the torso and arms group (95.9%) while the torso group (79.8%) was least effectively rashed. Although not formally presented, each of these experimental groups one-week post rash showed only about 5% area still remained rashed (of the total area wrapped); four subjects had no rash left at all, while 10 men showed less than 10% rashed area with the remaining subjects between 10 and 15%. The methods utilized for calculating the various rashed areas are as previously described (1,7).

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The average tolerance time for all subjects (n=24) on the 1st day of heat acclimatization (D_1) of 50.4 ± 1.8 min was significantly lower (P < 0.001) than the last (7th) day mean (D_7) of 99.3 ± 0.7 min, and there was no significant effect

(P > 0.05) due to groups (leg rash = 50.8 ± 3.7 min, 100 min; torso rash = 47.8 ± 1.4 min, 100 min; torso and arm rash = 55.0 ± 3.4 min, 97.9 ± 2.1 min; control = 44.2 ± 5.0 min, 100 min). Figure 1 contrasts the mean tolerance times between the three experimental and control groups during the last day of acclimatization (D_7) with the responses from the four test days (7, 14, 21 and 28 days after unwrapping of the three rashed groups). The average tolerance times after 7 (T_1) and 14 days (T_2) of unwrapping were significantly lower (P < 0.05) than during the last acclimatization day (D_7) and 28 days (T_4) of unwrapping; the 14th day responses (T_2) were also significantly lower than 21 day (T_3) values. There was no significant effect due to experimental group, although the control group as expected, displays little change in tolerance time throughout the periodic evaluations.

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During the heat acclimatization period, there were no significant differences (P > 0.05) due to experimental groups for mean body temperature ($\bar{T}_b$). There was a significant day by time effect (P < 0.05); at each 10-min time interval (0-40 min) the $\bar{T}_b$ responses ($^{\circ}$C) for the 1st acclimatization day (D_1) were higher than those for the last day (D_7). There were also significant differences (P < 0.05) amongst the time means within each day (D_1 and D_7). Figure 2 compares $\bar{T}_b$ between the three experimental rash groups (legs, torso, torso and arms) and the control group on the last day (D_7) of heat acclimatization, 7 days post rash (Test 1), 14 days post rash (Test 2), 21 days post rash (Test 3), and 28 days post rash (Test 4). In the analysis contrasting the $\bar{T}_b$ responses for the last acclimatization day with the four test days (0-50 min), the interaction of group by day by time was significant

$(P < 0.05)$. Since it is not feasible to calculate a critical difference $(cd_{.05})$ due to the number of possible comparisons as well as the disparity of group size, one can state that the trends across time by days differed among the four groups. Thus, there do not appear to be differences in $\bar{\bar{T}}_b$ between groups during the last acclimatization day. However, comparisons of $\bar{T}_b$ responses between groups during the post rash evaluations show higher values for all three rashed groups contrasted to the controls; this difference becomes more pronounced with time and persists for 21 days. At 28 days, the differences between groups seem to be mostly resolved. There do not appear to be major differences in the magnitude of response between the three rashed groups despite large group differences in the percent of total surface area rashed.

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Significant group differences were not found for body heat storage comparisons (Δ heat storage in kcal) between the 1st and last days of heat acclimatization. However, comparisons of heat storage values between the initial and last day of acclimatization show significantly higher values ($P < 0.01$) on the initial day at each time (0-40 min). Figure 3 illustrates the comparison of body heat storage values between the three experimental rash groups and the controls on the last day (D_7) of acclimatization and the four test days (7, 14, 21, and 28 days post rash). As was the case with $\bar{T}_b$, the triple order interaction (group by day by time) was significant for this heat storage analysis for responses from 10-50 min ($P < 0.05$). Again, one could assume statistically that the trends shown differ for groups by time and days. Therefore, it would seem that the mean heat storage values for all three

experimental rashed groups appear higher than control group values on test 1 (7 days post rash) while on test 2 (14 days post rash) only the legs rashed group seems demonstrably different from the controls. Distinct differences between these four groups for the last two test days (21 and 28 days post rash) appear speculative.

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Unexpectedly, the sweat rate analysis (kg/hr) showed a significantly higher ($P < 0.001$) rate per hour on the initial day of acclimatization (D_1) compared to the last day (D_7). There were no differences between the experimental groups. Figure 4 displays the sweat rate responses for the various groups, contrasting the responses on the last acclimatization day (D_7) with those for the four test days (T_1-T_4). The responses for the last day of acclimatization were significantly higher ($P < 0.01$) than those 7, 14 and 28 days (T_1, T_2, T_4) post rash; D_7 and T_3 (21 days post rash) did not differ significantly. Again, there is no effect due to experimental group. Clinical sweating observations comparing rashed areas to similar sites in controls were conducted immediately after rash induction, using the method of Tashiro et al. (14). The degree of sweating was estimated as (a) not different from control site, 1; (b) 50% of control site, 2; (c) borderline, scattered sweating, 3; or (d) no sweating, 4. The mean response ($\pm$SE) was 1.5 ± 0.2 for the controls compared to 2.6 ± 0.1 (legs group), 2.7 ± 0.1 (torso group), and 2.7 ± 0.2 (torso and arms group).

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Statistical analysis of heart rate responses showed no group differences. However, the mean response for the last acclimatization day was significantly lower (P < 0.05) than the response on the first day after 25 min of heat exposure. Figure 5 presents the HR responses for the various groups utilizing the same evaluation system shown previously. Although no statistically significant group differences were found, the torso group displays a trend for higher average responses throughout the test evaluations (7, 14, 21 and 28 days post rash).

DISCUSSION

Among the avenues for temperature regulation in men exposed to hot environments, the production and evaporation of sweat is the most important. The key feature in the pathophysiology of heat rash is the temporary dysfunction of the sweat glands which result in nonproduction of sweat (13). Thus, experimentally induced heat rash allows one to investigate a regulatory mechanism which, if damaged, can result in dangerous hyperthermia. Our previous work demonstrated that extensive experimentally induced heat rash (40 to 70% of the body surface) led to significant impairment in the capacity to perform work in hot environments (10). The present study demonstrates that this impairment exists when only 20 to 40% of the body surface is rashed. Seven days after the induction of the rash, 40% of the experimental rashed subjects (n=20) could not complete the 100-min exposure in the heat ($T_{re} \geq 39.5^{o}C$ and/or HR $\geq$ 180 bts/min), 70% of these same subjects were unsuccessful after 14 days, 35% after 21 days and 5% (one subject) after 28 days. These findings become more dramatic when one considers that all men were initially heat acclimatized.

Unfortunately, little information is available concerning the critical amount

of body surface area involvement with impaired sweating necessary to produce heat intolerance. Marked reductions in tolerance to heat were observed in both our previous study (10) involving heat rash (57% of average surface area rashed) and present study (32% surface area involved, n=20). However, physiological responses concerned with tolerance to heat of a burn patient (56% surface area covered with burned scar tissue) compared favorably with able-bodied controls (15). It was suggested that the patient (disabled 4 yrs) was able to compensate by producing a much greater sweat output (compensatory hyperhydrosis) from his available glands than normal. Although it is doubtful that this proposed compensatory mechanism played a major role in the regulatory responses of our rashed subjects, due to the short-term nature of the disorder, it is nevertheless possible that the similarity in the magnitude of physiological responses for the various rashed groups, despite large differences in area involvement could be some form of compensatory hyperhydrosis. Even the involvement of from 40% to less than 20% of the total surface area (16.7% for torso group) resulted in heat intolerance; although the average performance time of those rashed subjects was reduced by only 16.2 min compared to 32.3 min for our original group after 7 days, the reductions in performance time between studies become much closer after 14 days (24.2 min less in present study; 20.7 min, previous). Thus, short-term performance alterations may relate more closely to the percent of total area affected than longer-term. Tentatively, it would also seem that the critical surface area for reduced performance could be less than 20% of the total area.

The question of the critical surface area rashed for reduced performance may be linked to the percent surface area rashed relative to the sweating abundance for that particular area as well as to the degree of thermal stress associated with

different regional sweating responses. In general, sweating is greater on the head, neck and trunk than the extremities (3,7,11). However, during mild sweating the lower extremities may be the most important sites of high sweat loss, but during very profuse sweating the trunk and upper portions of the body (head and neck) become the more significant regions of sweat output (2,3). More specifically, the lower extremities tend to dominate sweating at lower temperatures (32-40°C) but sweating on the trunk increases more rapidly than that on the calf and thigh at higher temperatures; thus, the ratio of regional sweating to total sweating tended to approach unity at 45°C and to favor the trunk at higher temperatures (2,3). Since the experimental conditions for the present experiments (48.9°C, 20% rh) would seem to favor the importance of trunk regional sweating, this would help explain the near equality in elevated physiological responses for our rashed subjects (Fig. 2-3) despite large differences (20%) in the percent of total area rashed. Therefore, the critical surface area rashed which produces subsequent heat intolerance may be specific to the body region affected and the exact degree of environmental stress; in other words. for these as well as most clothing studies, a critical forcing function (i.e., combination of work level, temperature, humidity and air motion) may be essential to establish a given rash area as critical for heat intolerance.

Another modifying factor to consider in regional heat rash is the state of acclimatization. Hofler (5) and more recently Shvartz et al. (12) have independently found lower sweat rates over the legs than the trunk prior to heat acclimatization, but a larger increase in limb than in trunk sweat rate after acclimatization. These authors imply that the smaller increase in chest than limb sweat rate during acclimatization may be related to the greater efficiency for heat exchange over

the chest than over the limbs (12), or higher limb sweating after acclimatization could mean a better utilization of body surface for evaporation (5). It is known from convective heat loss considerations, and particularly the air motion generated by walking, that evaporative coefficients are higher on the limbs than on the trunk (6). Thus, prior acclimatization of our rashed subjects would seem to aid the torso, and torso and arms groups more than the legs group and help to possibly offset the loss of trunk regional sweating in this particular environment. We cannot explain why adding some of the surface area of the arms (torso-arms group) to the rashed area did not result in a reduction in response contrasted to the torso group. Possibly the upper extremities are very inefficient areas for heat exchange at least for this experimental situation, or, again, perhaps we were not at a critical forcing function to display the effect of the added area.

From our observations of reduced tolerance time and elevated physiological responses of the three rash groups, it appears that some degree of heat intolerance persists for about three weeks and subsides by four weeks. Once again, while there was a dramatic reduction in ability to work in the heat, coupled with demonstrable elevations in body temperature and calorie storage, the hypohidrotic skin areas of the rashed subjects were not markedly abnormal in appearance. This finding on the persistence of heat intolerance associated with heat rash agrees very closely with the conclusions of Griffin et al. (1) who found clinical depression of sweating up to 21 days with a return to the prerashed state by 28 days in a similar subject population. However, the particular race of test subjects may influence the degree and persistence of rash. Hindson (4) reports over 90% of British troops developed experimentally produced heat rash and hypohydrosis while, utilizing the same techniques, only 45% of Gurkha troops developed the rash but 88% had detectable hypohidrosis; persistence also seemed affected by the particular race.

In summary, the critical amount of surface area for heat intolerance from heat rash appears to be related to the specific region of the body and associated sweating responses; smaller rashed areas of the trunk because of greater potential for sweating in abundance, may produce responses to heat stress comparable to those of larger rashed areas of the limbs. The specific environmental conditions and state of acclimatization are important considerations when considering the effects of regional heat rash. The elevated physiological responses and associated heat intolerance of these rashed subjects appears to persist to some degree for 21 days but becomes resolved by 28 days. Thus, small but specific regional area rashing can produce significant heat intolerance in man.

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TABLE 1. Comparison of rashed areas, effectiveness of rashing and percent of the total areas rashed between various experimental rash groups.

Body Area	Rashed Subj	Total Area Wrapped, m^2	Non-rashed Area*, m^2	Rashed Area, m^2	Rashed,% Wrapped	Total Surface Area,m^2	%Total Area Rashed
Legs	A	.7398	.0973	.6425	87	1.87	34
	B	.8211	.0667	.7544	92	1.89	40
	C	.8969	.0366	.8603	96	1.91	45
	D	.8601	.0411	.8190	95	1.97	41
	E	.8524	.0833	.7691	90	1.88	41
	F	.8601	.0205	.8396	98	1.98	42
$\bar{X}$		.8384	.0576	.7808	93.0	1.92	40.5
$\pm$SE		.0220	.0121	.0322	1.7	0.02	1.5
Torso	A	.3312	.1442	.1870	56	1.70	11
	B	.4295	.0386	.3909	91	2.03	19
	C	.4602	.0591	.4011	87	1.95	21
	D	.4050	.0443	.3607	89	1.79	20
	E	.3295	.1161	.2134	65	1.80	12
	F	.3714	.0327	.3387	91	1.99	17
$\bar{X}$		.3878	.0725	.3153	79.8	1.88	16.7
$\pm$SE		.0217	.0189	.0377	6.3	0.05	1.7
Torso & Arms	A	.6869	.0430	.6439	94	1.75	37
	B	.8792	.0048	.8744	99	1.99	44
	C	.6959	.0060	.6899	99	1.82	38
	D	.7150	.0555	.6595	92	1.95	34
	E	.7493	.0478	.7015	94	1.96	36
	F	.6802	.0146	.6656	98	1.75	38
	G	.6656	.0137	.6519	98	1.83	36
	H	.6868	.0465	.6403	93	1.73	37
$\bar{X}$		.7199	.0290	.6909	95.9	1.85	37.5
$\pm$SE		.0245	.0075	.0273	1.0	0.04	1.0
Control	A					1.97	
	B					1.97	
	C					1.83	
	D					1.86	
$\bar{X}$						1.91	
$\pm$SE						0.04	

*The non-rashed areas were calculated and the rashed areas obtained by subtraction from the total wrapped area.

FIGURE LEGENDS

FIG. 1. Differences in tolerance time between the experimental rash groups (legs, torso and arms-torso) and control group while walking (1.56 m s^{-1}) in the heat (48.9°C, 20% rh) for an attempted 100 min. Comparisons are made between the last (D$_7$) day of heat acclimatization, and seven (T$_1$), fourteen (T$_2$), twenty-one (T$_3$) and twenty-eight (T$_4$) days after unwrapping the experimental groups.

FIG. 2. Comparisons between the various experimental rash groups and control group for mean body temperature (°C) on the final day of heat acclimatization, and 7, 14, 21 and 28 days post rash.

FIG. 3. Comparisons between the various experimental rash and control groups for body heat storage (kcal) on the final day of heat acclimatization, and 7, 14, 21 or 28 days post rash.

FIG. 4. Differences in sweat rate (kg/hr) between the various experimental rash and control groups while walking (1.56 m s^{-1}) in the heat (48.9°C, 20% rh) for an attempted 100 min. Comparisons are made between the last (D$_7$) heat acclimatization day, and 7 (T$_1$), 14 (T$_2$), 21 (T$_3$) and 28 (T$_4$) days post rash.

FIG. 5. Comparisons between the various experimental rash and control groups for heart rate (bts/min) on the final day of heat acclimatization, and 7, 14, 21 or 28 days post rash.

FIG. 1

FIG.2

TEST 2
[14 DAYS POST RASH]

△ HEAT STORAGE (kcal)

TEST 1
[7 DAYS POST RASH]

△ HEAT STORAGE (kcal)

DAY 7
[FINAL DAY OF ACCLIMATIZATION]

△ HEAT STORAGE (kcal)

TEST 4
[28 DAYS POST RASH]

△ HEAT STORAGE (kcal)

TEST 3
[21 DAYS POST RASH]
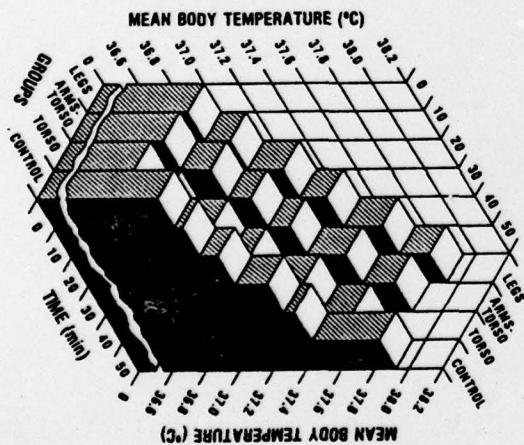
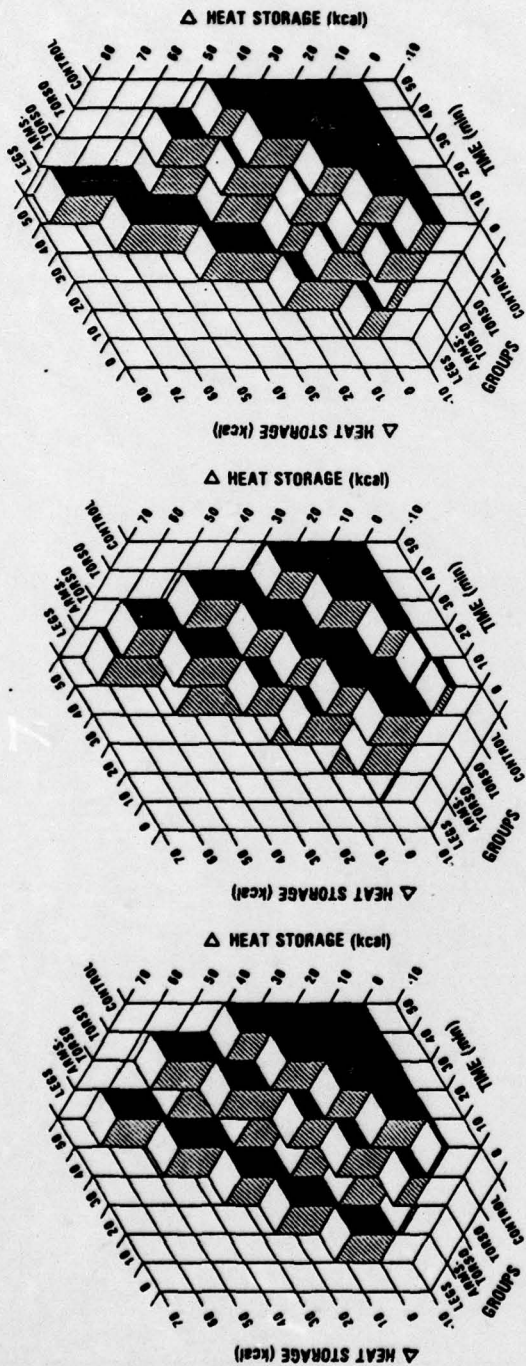△ HEAT STORAGE (kcal)
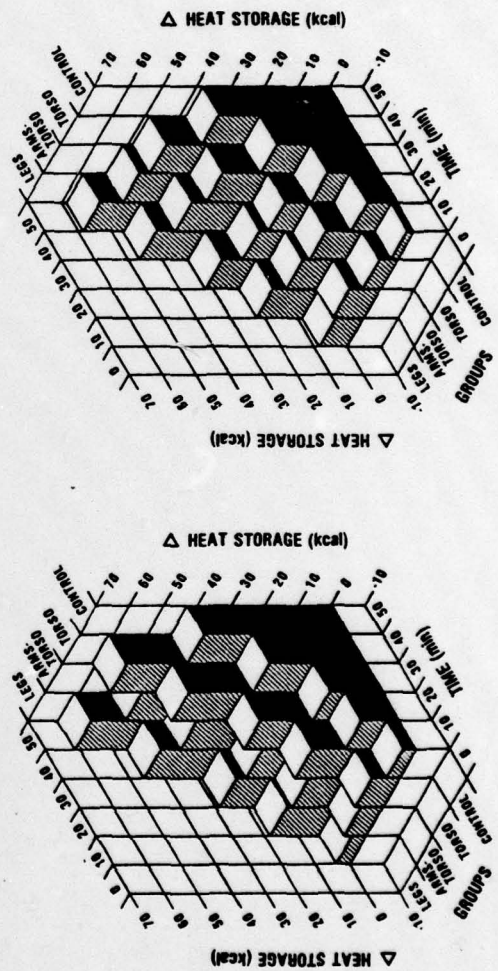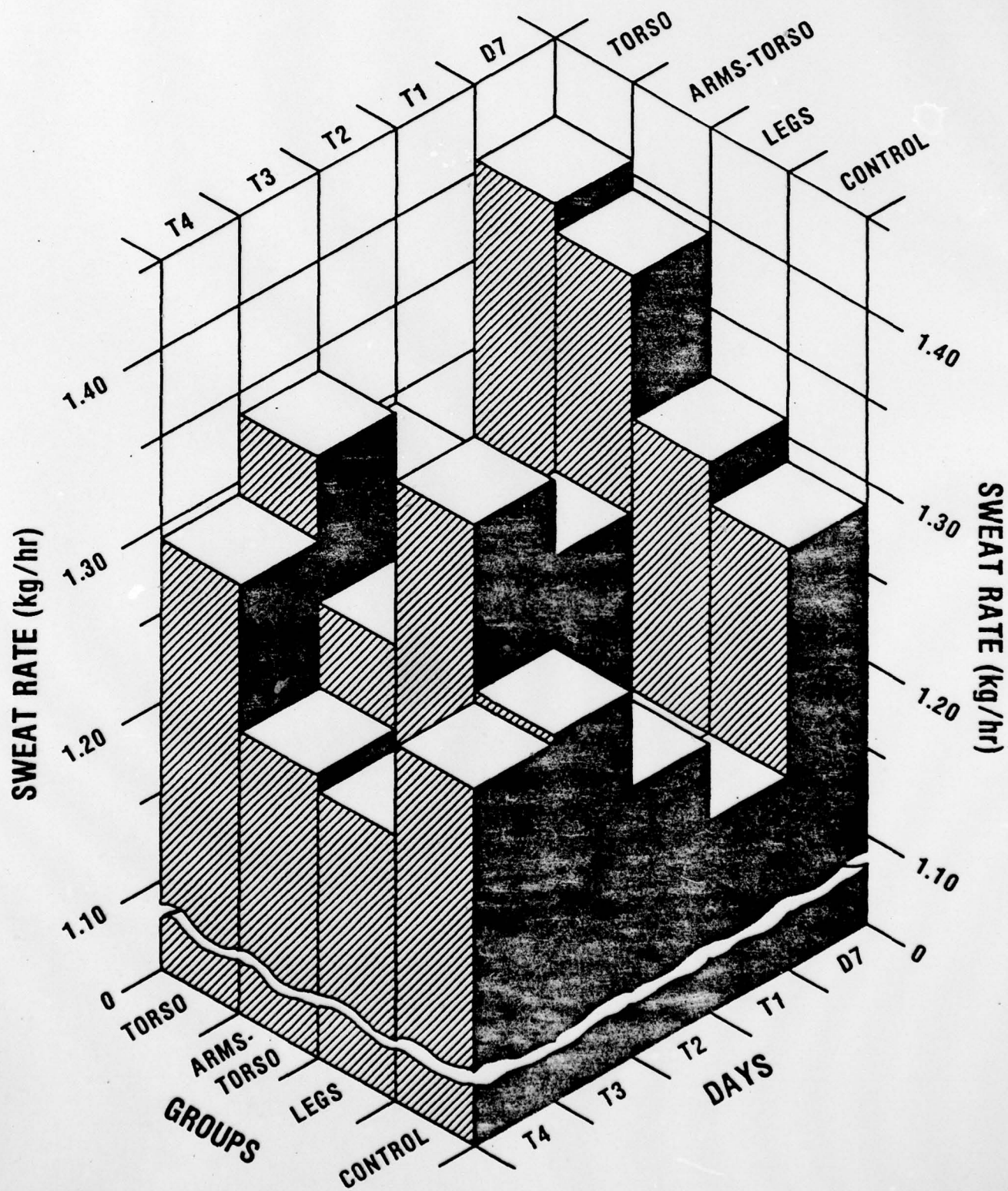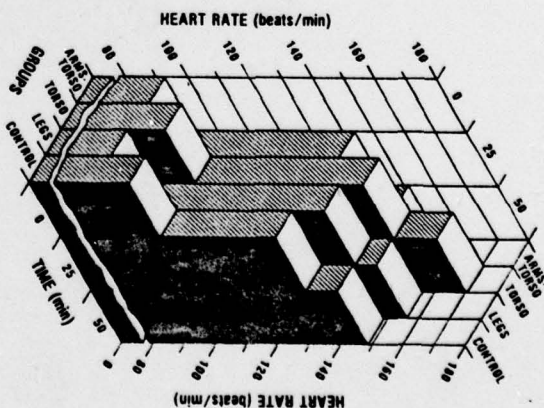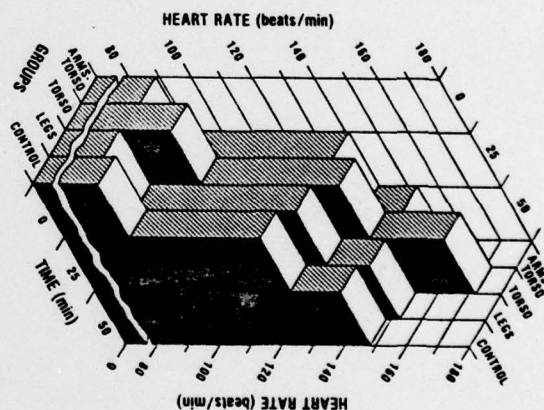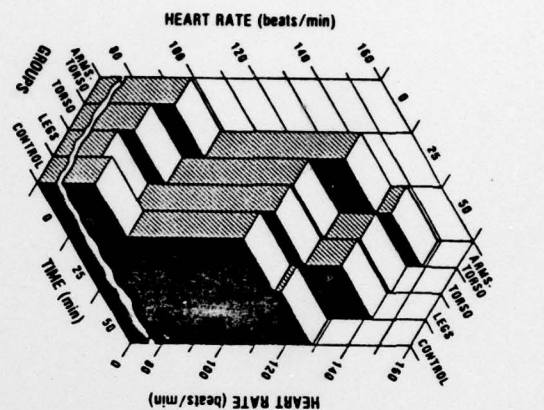
FIG. 3

FIG.4

HEART RATE (beats/min)
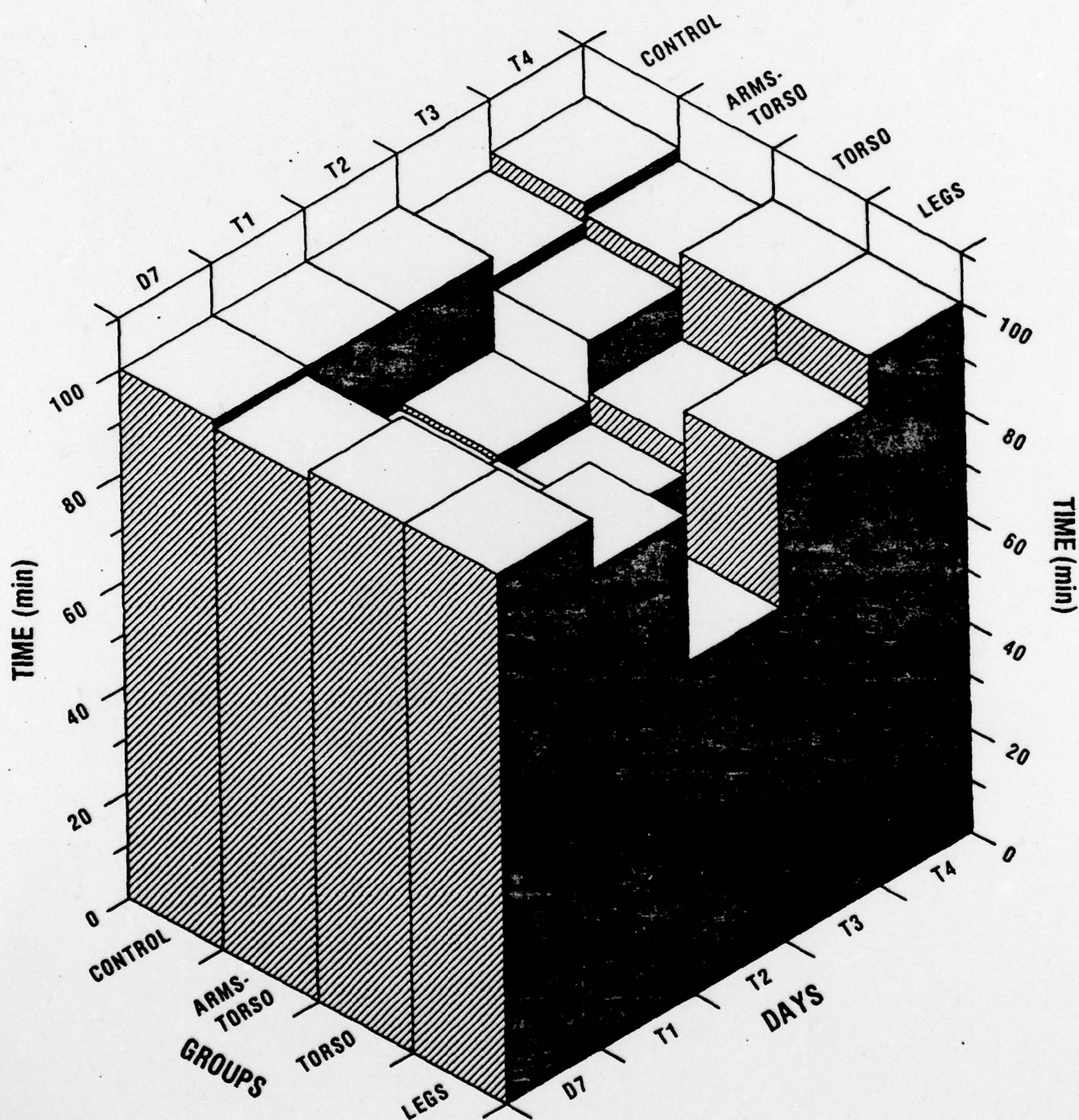
DAY 7
[FINAL DAY OF ACCLIMATIZATION]
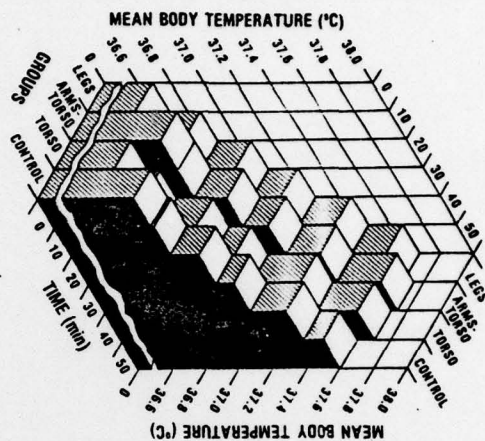
TEST 1
[7 DAYS POST RASH]

TEST 2
[14 DAYS POST RASH]

HEART RATE (beats/min)
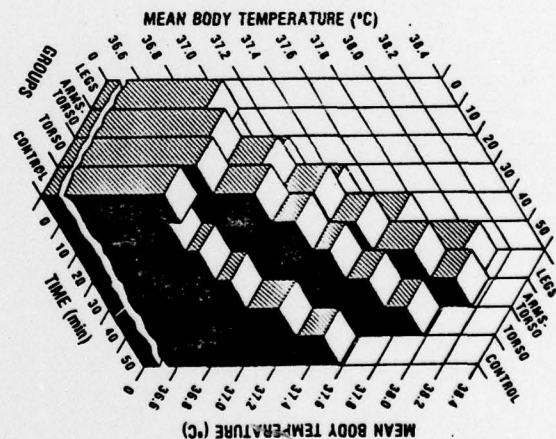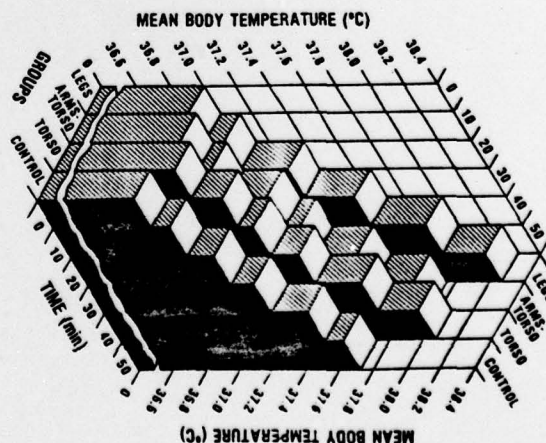
TEST 3
[21 DAYS POST RASH]

TEST 4
[28 DAYS POST RASH]

FIG. 5